Chronic myeloproliferative diseases with and without the Ph chromosome: some unresolved issues.

Journal: Leukemia

Publication Year: 2009

Authors: J M Goldman, A R Green, T Holyoake, C Jamieson, R Mesa, T Mughal, F Pellicano, D Perrotti, R Skoda, A M Vannucchi

PubMed link: 19641523

Funding Grants: Derivation and Characterization of Myeloproliferative Disorder Stem Cells from Human ES Cells, Derivation and Characterization of Cancer Stem Cells from Human ES Cells

Public Summary:

Scientific Abstract:
Ph-positive chronic myeloid leukemia (CML) and Ph-negative chronic myeloproliferative diseases (MPDs), characterized in many cases by the presence of the JAK2(V617F) mutation, have many features in common and yet also show fundamental differences. In this review, we pose five discrete and related questions relevant to both categories of hematological malignancy, namely: What are the mechanisms that underlie disease progression from a relatively benign or chronic phase? By what therapeutic methods might one target residual leukemia stem cells in CML? Is JAK2(V617F) the original molecular event in MPD? What epigenetic events must have a role in dictating disease phenotype in MPDs? And finally, Will the benefits conferred by current or future JAK2(V617F) inhibitors equal or even surpass the clinical success that has resulted from the use of tyrosine kinase inhibitors in CML? These and others questions must be addressed and in some cases should be answered in the foreseeable future.